Patterns of atrial activation during right ventricular pacing in patients with concealed left-sided Kent pathways

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SUMMARY A 'concealed' accessory pathway was suspected in 12 patients because of eccentric left atrial activation during tachycardia. Retrograde conduction during ventricular pacing may occur over the atrioventricular node, the accessory pathway, or both. There were 4 patterns of ventriculoatrial conduction in response to ventricular extrastimuli (V_2) at various coupling intervals: (1) exclusive accessory pathway conduction throughout the cardiac cycle in 2 patients; (2) exclusive accessory pathway conduction at long coupling intervals and exclusive atrioventricular node conduction at short coupling intervals in 2 patients; (3) variably fused accessory pathway/atrioventricular node conduction at long coupling intervals but exclusive accessory pathway conduction at short coupling intervals in 4 patients; (4) fused accessory pathway/atrioventricular node conduction at long coupling intervals but exclusive atrioventricular node conduction at short coupling intervals in 4 patients.

With increased prematurity of V_2 the ventricle to right atrial interval prolonged conspicuously in 11 of 12 patients whereas the ventricle to left atrial interval remained constant until the refractory period of the accessory pathway in all but 2 instances where intraventricular delay occurred. This study emphasises the importance of left atrial recordings in these patients.

It is now well recognised that concealed ventriculo-atrial anomalous pathways provide the anatomical substrate for a large proportion of paroxysmal re-entrant tachycardias (Slama *et al.*, 1973; Spurrell *et al.*, 1974; Rowland *et al.*, 1977). These pathways are designated 'concealed' because they do not conduct anterogradely and therefore there are no electrocardiographic signs of ventricular pre-excitation. They do, however, produce atrial pre-excitation in response to ventricular stimulation. The degree of atrial pre-excitation via a concealed accessory pathway is dependent upon tissue refractoriness and conduction times from the point of ventricular stimulation to the atrial recording site. Constancy of the ventriculoatrial conduction time has been used as a criterion for conduction in an anomalous ventriculoatrial pathway, but evidence in patients with overt Wolff-Parkinson-White syndrome (Tonkin *et al.*, 1975; Sung *et al.*, 1977a) suggests that this criterion taken by itself may result in underdiagnosis of concealed ventriculoatrial pathways.

This study was designed to investigate retrograde conduction patterns in patients with concealed ventriculoatrial pathways.

Patients and methods

The 12 patients were admitted for investigation of recurrent paroxysmal tachycardias. Clinical details are summarised in Table 1. In all patients physical examination, chest radiography, and routine haematological investigations were normal. In 5 patients the PR interval was short and in 7 patients it was normal. The QRS complex during sinus rhythm was normal in all patients.

All patients were studied in the post-absorptive, non-sedated state and medications were discontinued at least 72 hours before study. Under local anaesthetic, 4 bipolar USCI electrode catheters with 1 cm interelectrode distance were introduced via the femoral vein and positioned in the right ventricular apex, against the septal leaflet of the tricuspid valve and in the high right atrium (2 catheters). A quadripolar USCI catheter with 1 cm interelectrode distances, was inserted via the left

Table 1

Case no.	Age, sex	symptoms	ECG PR (s)	QRS	Documented arrhythmias
1	48, M	Palpitation	0·20	N	SVT
2	40, F	Palpitation and dizziness	0·11	N	SVT, AF
3	21, F	Palpitation	0·12	N	SVT
4	50, M	Palpitation	0·18	N	SVT, AF
5	41, M	Palpitation	0·12	N	SVT
6	47, F	Palpitation	0·20	N	SVT
7	39, M	Palpitation	0·19	N	SVT
8	54, F	Palpitation	0·21	N	SVT
9	40, F	Palpitation	0·20	N	SVT
10	47, M	Palpitation	0·12	N	SVT
11	63, M	Palpitation	0·11	N	SVT, AF
12	15, F	Palpitation	0·16	N	SVT

N, normal; SVT, supraventricular tachycardia; AF, atrial fibrillation.

basilic vein and positioned in the coronary sinus. Electrograms from the intracardiac catheters were recorded on a 16-channel Elema Mingograf recorder at a paper speed of 100 mm/s together with surface electrocardiographic leads I, aVF, V1, and V6. Intracardiac signals were filtered between 50 and 700 Hz. Stimulation was performed using a Devices 4270 isolated stimulator with pulse widths of 1·5 to 2 ms at a voltage approximately twice the diastolic threshold.

In all patients anterograde and retrograde conduction studies were performed using the extra-stimulus technique (Durrer *et al.*, 1967). Retrograde conduction was studied during right ventricular apical pacing. The ventriculoatrial conduction times were measured from the onset of ventricular activation to the earliest clearly distinguishable atrial electrogram in the right atrium (V-RA) and coronary sinus (V-LA). The earliest point of atrial activation during sustained tachycardia was taken as indicating the approximate location of the accessory pathway. This point was determined by coronary sinus withdrawal mapping. The atrial activation sequence during tachycardia was considered to represent atrial depolarisation by exclusive conduction in the accessory pathway (Fig. 1).

Rapid atrial pacing at right and left atrial sites was performed in all patients. In 7 patients atrial fibrillation was induced electively. Anterograde
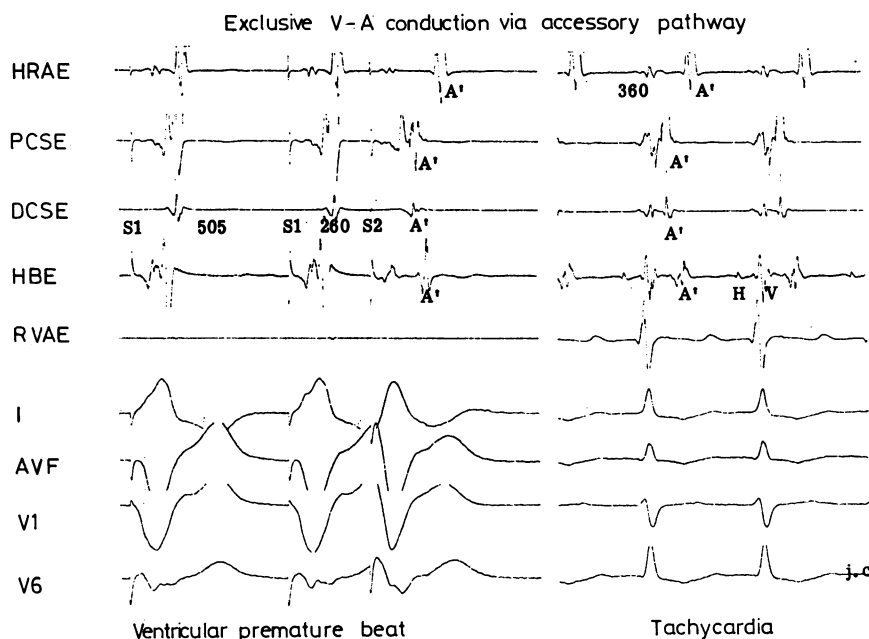

Fig. 1 *Comparison of atrial activation sequence after a ventricular premature beat* (left panel) *and during tachycardia* (right panel) *in case 3 (group 3). A ventricular premature stimulus (S₂) at 260 ms coupling interval after a regular paced sequence (S₁-S₁) results in retrograde atrial activation (A') identical to that seen during re-entrant tachycardia. HRAE, high right atrial electrogram; PSCE, proximal coronary sinus electrogram; DCSE, distal coronary sinus electrogram; HBE, His bundle electrogram; RVAE, right ventricular apex electrogram (not recorded during ventricular pacing). I, AVF, V1, V6 = surface electrocardiographic leads.*

conduction in the accessory pathway was excluded by the lack of pre-excitation in response to atrial stimulation near the accessory pathway (Boineau *et al.*, 1973; Denes *et al.*, 1977) and in 7 patients by the absence of pre-excitation during atrial fibrillation. Retrograde conduction curves were obtained by plotting the values of V-LA and V-RA and the corresponding intervals between successive atrial electrograms in response to the premature stimulus V_2 against the coupling interval V_1-V_2 of the extrastimulus.

Results

The results are summarised in Table 2.

In all 12 patients re-entrant atrioventricular tachycardia was initiated by a single atrial premature stimulus. In 5 patients a right ventricular apical stimulus was also effective in initiating tachycardia. Earliest atrial activation during tachycardia was in the coronary sinus recording in all 12 patients: proximal coronary sinus in 7 patients and distal coronary sinus in 5 patients. The low right atrial electrogram could be clearly seen during tachycardia in all 12 patients and followed the earliest atrial electrogram in the coronary sinus region.

Retrograde atrial activation patterns during right ventricular pacing and programmed extrastimulation were of 4 different types (Table 2).

GROUP 1
In this group of 2 patients, the ventriculoatrial intervals remained constant at all coupling intervals $(V_1$-$V_2)$ of ventricular extrastimuli (V_2). In 1 patient there was a small but equal increase in the V-LA and V-RA conduction times at very short coupling intervals. The relation between the right and left atrial electrograms did not change. In both patients tachycardia was initiated by a
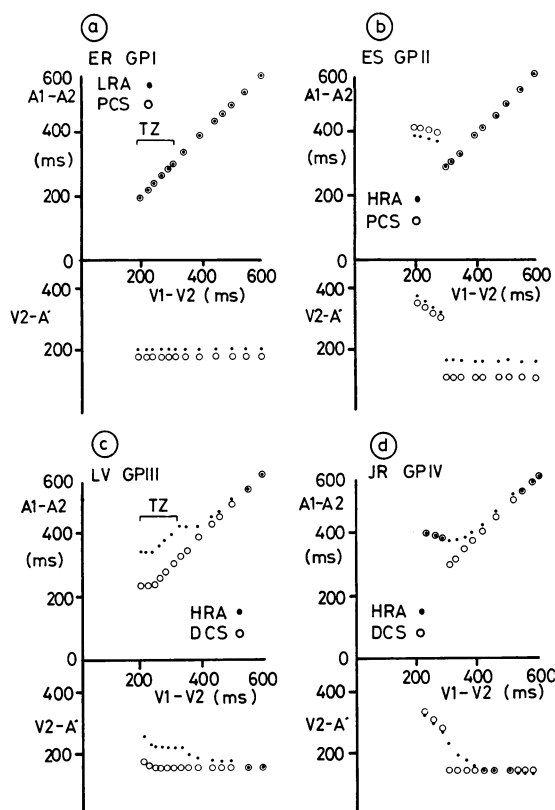

Fig. 2 *Retrograde conduction curves from patients representing each of the 4 groups. For discussion see text. LRA, low right atrial electrogram; HRA, high right atrial electrogram; PCS, proximal coronary sinus electrogram; DCS, distal coronary sinus electrogram; TZ, tachycardia zone.*

programmed atrial or ventricular premature beat. The atrial activation sequence during tachycardia was identical to that observed during programmed ventricular extrastimulation. The retrograde conduction curve from one of these patients is shown in Fig. 2a.

GROUP 2
In 2 patients, the V-LA and V-RA conduction intervals remained more or less constant at long coupling intervals of V_2. At shorter coupling intervals there was a sudden increase in the V-LA and V-RA intervals. At all values of V_1-V_2 shorter than that which resulted in the sudden increase in the ventriculoatrial intervals, there was a progressive smooth increase in the ventriculoatrial conduction times without a further change in the relation of the atrial electrograms (Fig. 2b). In this group,

Table 2

Case no.	Site of AP	Mode of initiation of tachycardia	Pattern of atrial activation (VPBs)	Group
1	Distal CS	APB	F →AVN	4
2	Distal CS	APB, VPB	F →AP	3
3	Proximal CS	APB	F →AP	3
4	Proximal CS	APB	AP→AVN	2
5	Distal CS	APB	F →AVN	4
6	Proximal CS	APB	AP→AVN	2
7	Proximal CS	APB, VPB	AP→	1
8	Proximal CS	APB, VPB	F →AP	3
9	Distal CS	APB	F →AVN	4
10	Proximal CS	APB	F →AVN	4
11	Proximal CS	APB, VPB	AP→	1
12	Distal CS	APB	F →AP	3

AP, accessory pathway; CS, coronary sinus; AVN, AV nodal; F, fusion; APB, programmed atrial premature beat; VPB, programmed ventricular premature beat.

tachycardia could not be initiated by a ventricular premature stimulus but was reliably initiated by a single atrial premature stimulus. In 1 of these patients the atrial activation sequence during tachycardia was identical to that observed with premature beats in the range of coupling intervals which resulted in constant ventriculoatrial intervals. In the other patient (case 4), the recordings were difficult to interpret partly because the low right atrial electrogram was not clearly seen. These recordings are shown in Fig. 3 and the results presented graphically in Fig. 4.

GROUP 3
In 4 patients various degrees of atrial fusion (V-LA and V-RA not changing concordantly) were seen in response to longer coupling intervals of V_2. As the coupling interval of V_2 was reduced, the V-RA

interval increased gradually but the V-LA interval remained constant. Further decreases in the V_1-V_2 interval resulted in an attenuation of the increase in V-RA time which gradually plateaued, forming a curve parallel to the V-LA curve (Fig. 2c). The V-LA interval remained constant throughout in 2 patients, with a small increase in V-LA and V-RA intervals at very short coupling intervals in 1 patient. In case 3 this basic pattern was modified (Fig. 5). At coupling intervals greater than 360 ms (Fig. 5 and 6) low right atrial activation was progressively delayed as V_1-V_2 was decreased. At 360 ms, there was a sudden increase in the V-RA interval which remained constant with coupling intervals, V_1-V_2 between 360 and 320 ms. The V-LA interval remained unchanged. At V_1-V_2 of 300 ms the V-RA interval shortened again and increased as V_2 become more premature. Coupling
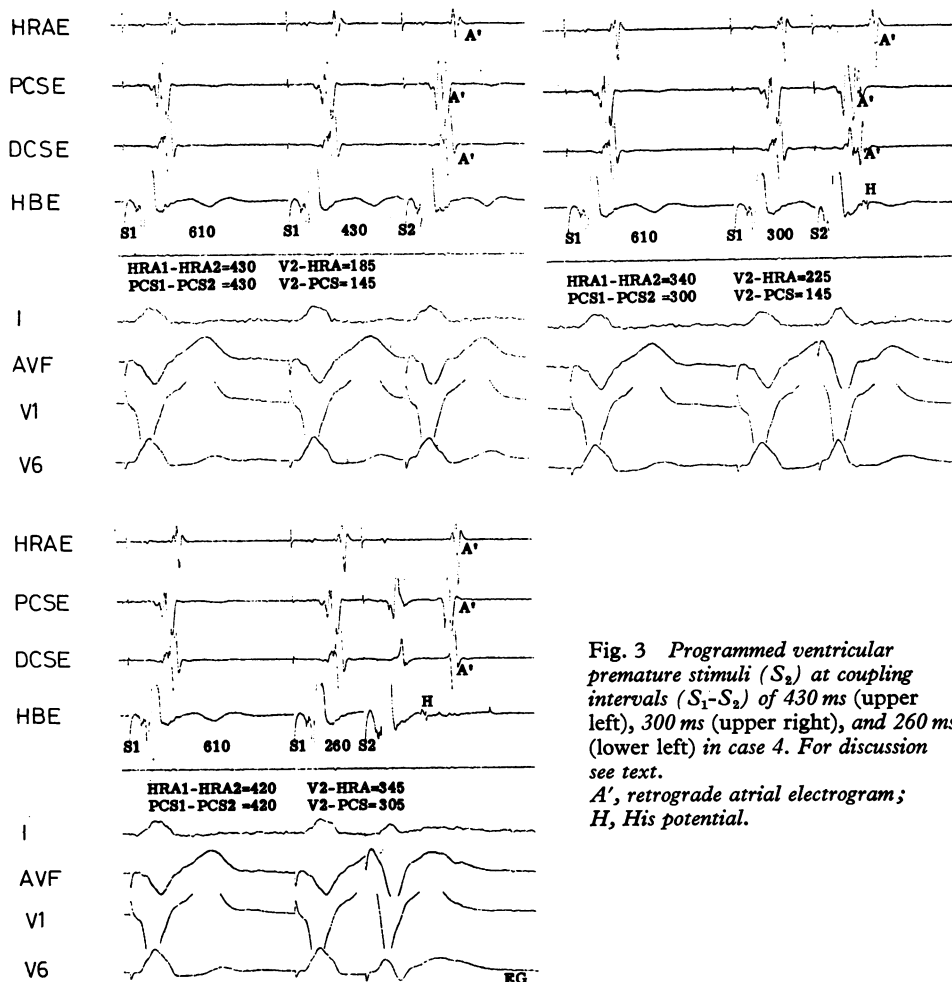

Fig. 3 *Programmed ventricular premature stimuli (S₂) at coupling intervals (S₁-S₂) of 430 ms (upper left), 300 ms (upper right), and 260 ms (lower left) in case 4. For discussion see text.*
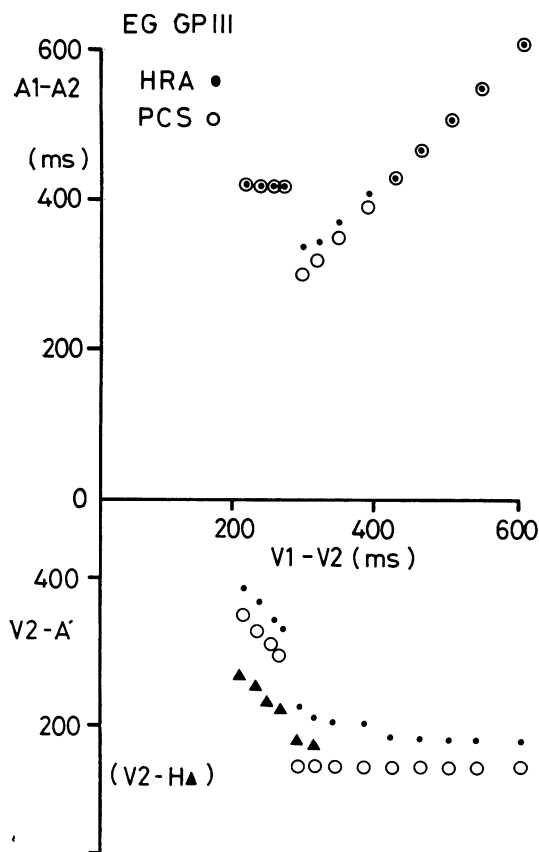*A', retrograde atrial electrogram; H, His potential.*

Fig. 4 *Retrograde conduction curve of case 4 (group 2).* *For clarity H$_1$-H$_2$ intervals are omitted; the V-H* *interval is plotted on the lower part of the graph. For* *discussion see text.*

intervals of V$_2$ shorter than 260 ms did not result in any further increase in the V-RA interval (Fig. 5).

In all 4 patients in this group, re-entrant tachycardia could be initiated by a single atrial premature beat but a single ventricular extrastimulus was effective in only 3 patients. The atrial activation sequence during tachycardia was identical to that observed at coupling intervals of V$_2$ which resulted in parallel V-RA and V-LA curves.

GROUP 4
In the 4 patients in this group, atrial fusion was also observed at longer coupling intervals of V$_2$. As V$_1$-V$_2$ was decreased the V-RA interval gradually increased while the V-LA interval remained constant. At a shorter coupling interval, there was a sudden increase in the V-LA interval. Values of V$_1$-V$_2$ shorter than that which resulted in the

sudden increase produced a progressive concordant increase in V-LA and V-RA intervals (Fig. 2d).

Tachycardia could be initiated by atrial extrastimuli but ventricular premature beats were ineffective. The atrial activation sequence during tachycardia was different from that observed in response to a ventricular extrastimulus at any coupling interval.

Discussion

Four distinct patterns of retrograde conduction were observed in this series of patients. These basic patterns are determined by the relative contribution to atrial activation over the normal and accessory pathways.

(1) Patients in group 1 showed constancy of the ventriculoatrial intervals at all coupling intervals of V$_2$ as a result of predominant conduction in the accessory pathway (Fig. 2a) (Narula, 1974; Wellens and Durrer, 1974). The small but equal increase in V-RA and V-LA intervals in 1 patient was attributed to intraventricular delay.

(2) Conduction over the accessory pathway predominated at longer coupling intervals of V$_2$ in patients in group 2. When the V$_1$-V$_2$ interval was reduced to within the retrograde effective refractory period of the accessory pathway the sudden increase in V-RA and V-LA intervals and altered atrial activation sequence reflects continued conduction in the normal pathway (Fig. 2b).

(3) Conduction over both the normal and accessory pathways resulting in atrial fusion at long coupling intervals of V$_2$ was observed in patients in group 3 (Fig. 2c). Conduction in the accessory pathway results in a constant V-LA interval throughout, while delayed conduction in the normal pathway results in a gradual increase of the V-RA interval.

While this delay exceeds the conduction time from the accessory pathway to the right atrium (at the site of recording) the V-RA interval parallels the V-LA interval. In 2 patients the beginning of the V-RA 'plateau' is coincident with the upper limit of the tachycardia zone suggesting that block rather than delay in the normal pathway allowed capture of the right atrium as a result of spread of activation from the accessory pathway.

(4) Atrial fusion was observed at all coupling intervals of V$_2$ until block in the accessory pathway in patients in group 4 (Fig. 2d and Fig. 7). The gradual increase in the V-RA interval as V$_1$-V$_2$ was increased indicates atrial activation as a result of conduction over the normal pathway. In contrast, the V-LA interval remains constant until block occurs in the accessory pathway. Continued conduc-
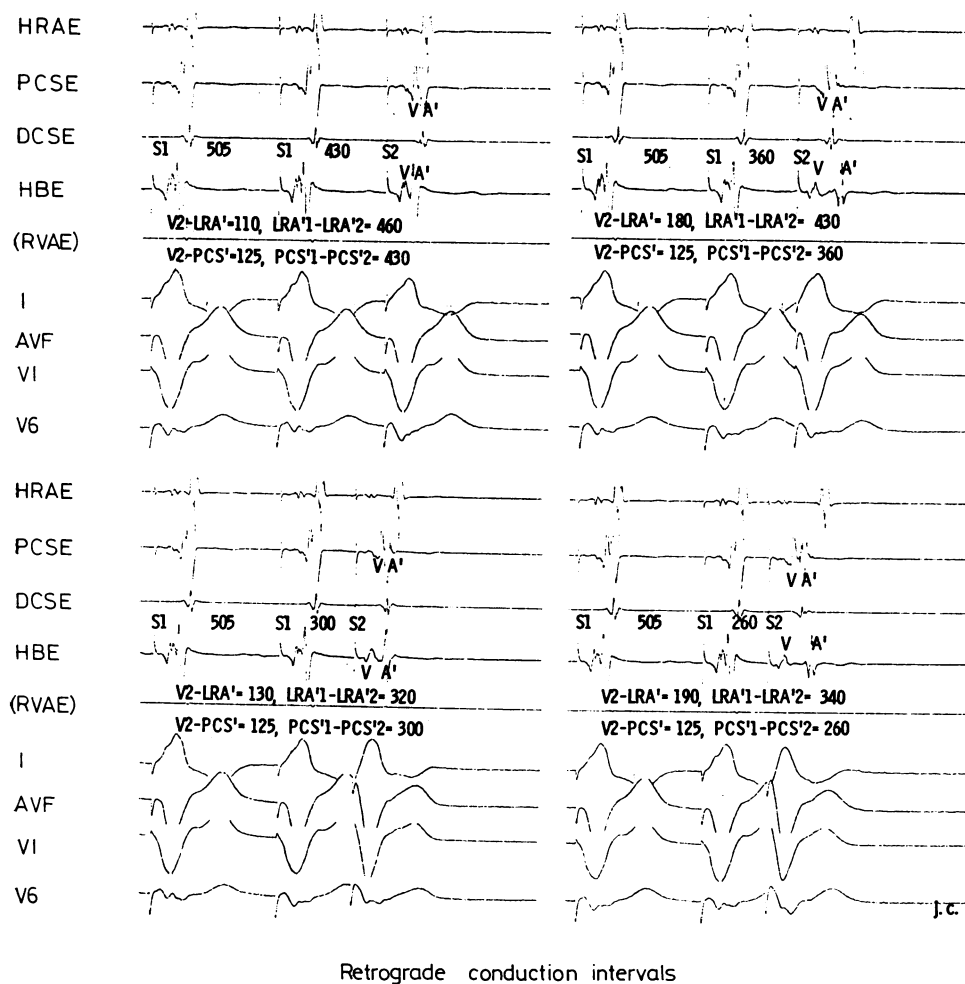
Retrograde conduction intervals

Fig. 5   *Programmed ventricular premature stimuli (S₂) at coupling intervals of 430 ms (upper left), 360 ms (upper right), 300 ms (lower left), and 260 ms (lower right) in case 3. For discussion see text.*

tion in the normal pathway results in a sudden jump in the V-LA interval associated with a change in atrial activation sequence (Tonkin *et al.*, 1975). Exclusive accessory pathway conduction, therefore, never occurred in this group. Two patients, one from each of group 2 and group 3 showed conduction patterns which appeared to represent a modification of the basic conduction pattern of the group. In case 4 the slight increase in the V-RA interval between coupling intervals of V_2 of between 430 and 300 ms was attributed to intra-atrial delay. The absence of any severe changes in atrial activation sequence throughout the curve (Fig. 3) is not unexpected because the accessory pathway was sited near the atrioventricular node in

this patient and conduction in either the normal or accessory pathways may result in similar retrograde activation sequences (Gallagher *et al.*, 1977). However, because the low right atrial electrogram was not clear, the complete atrial activation sequence is not known.

Case 3 had a variation of the basic pattern of group 3 (Fig. 5 and 6). It is postulated that the sudden delay in the right atrial electrogram, sustained over a small range of coupling intervals, is a result of failure of ventriculoatrial conduction in the normal pathway, allowing capture of the right atrium by continued conduction in the accessory pathway. This interpretation is supported by the observation that the V-RA intervals in this zone
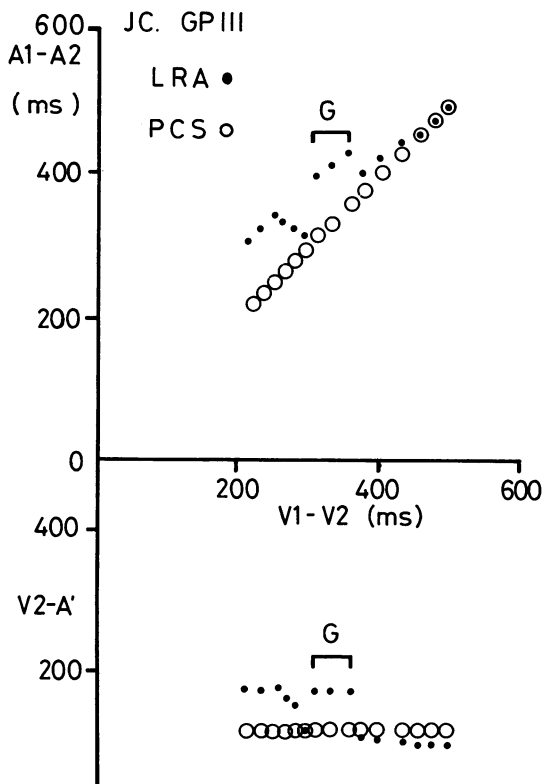
Fig. 6 *Retrograde conduction curve from case 3 (group 3). For discussion see text. G, gap in retrograde His Purkinje/atrioventricular nodal conduction.*

are similar to those seen at short coupling intervals of V_2 when accessory pathway conduction predominates. In effect, there is a gap in ventriculoatrial conduction in the normal pathway (Akhtar et al., 1974) but this gap is concealed by continued conduction in the accessory pathway. A similar case has been described by Wellens (1977).

Svenson et al. (1975) studied patterns of ventriculoatrial conduction in patients with overt Wolff-Parkinson-White syndrome and found fusion patterns only in patients with lateral bypass tracts. In the 5 patients in our series with accessory pathways in the region of the distal coronary sinus, atrial fusion was demonstrated at long coupling intervals of V_2, with exclusive accessory pathway conduction at short coupling intervals in 2 patients and exclusive atrioventricular nodal conduction at short coupling intervals in 3 patients. In contrast to the observation of Svenson et al. (1975) we showed atrial fusion patterns in 3 of the 7 patients with proximally located bypass tracts, 2 of whom showed exclusive bypass tract conduction only at

short coupling intervals of V_2. Of the other 4 patients with proximally located pathways, 2 disclosed predominant accessory pathway conduction throughout (group 1) and 2 had the pattern typical of group 2.

Of the 5 patients with short PR intervals, 4 patients had atrial fusion patterns at long coupling intervals of V_2 and 1 patient showed exclusive accessory pathway conduction throughout. Retrograde conduction in an atrionodal pathway may result in earliest atrial activation in the region of the proximal coronary sinus (Camm et al., 1978). In 2 patients this possibility was excluded by the finding of earliest atrial activity in the distal coronary sinus electrogram. In the 3 other patients with proximally situated bypass tracts a ventricular premature stimulus delivered at the time of the His bundle potential resulted in exact atrial capture. This observation excludes retrograde conduction in an atrionodal pathway (Ward et al., 1978) and confirms ventriculoatrial conduction in a direct ventriculoatrial connection (Coumel and Attuel, 1974; Zipes et al., 1974; Neuss et al., 1975; Sellers et al., 1976).

Tachycardia could not be initiated by a ventricular premature beat in any patient in whom there was evidence of normal pathway ventriculoatrial conduction at all coupling intervals of V_2 (group 2 and group 4). It is suggested that re-entry towards the ventricles over the atrioventricular node-His pathway was prevented by continued retrograde conduction in this pathway. Ventricular premature beats were effective in initiating tachycardia in 2 of the 4 patients who showed exclusive accessory pathway conduction only at short coupling intervals (group 3). In these patients, the upper limit of the
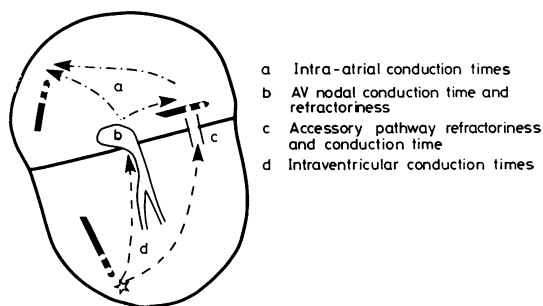

	a	Intra-atrial conduction times
	b	AV nodal conduction time and refractoriness
	c	Accessory pathway refractoriness and conduction time
	d	Intraventricular conduction times

Fig. 7 *Determinants of retrograde conduction patterns during right ventricular extrastimulation in patients with accessory bypass tracts. The pattern of atrial activation is determined by the relative conduction times from the site of stimulation to the site of recording in both the normal and abnormal pathways and the state of refractoriness in these pathways.*

tachycardia zone coincided with the upper limit of the V-RA interval plateau. The inability of ventricular premature beats to initiate tachycardia in the other 2 patients in group 3 may be related to concealed retrograde invasion of the normal pathway at short coupling intervals (Wellens, 1977). These observations are similar to those made by Sung *et al.* (1977a) in patients with overt type A Wolff-Parkinson-White syndrome.

In the absence of accessory ventriculoatrial connections, atrial activation during ventricular pacing begins close to the atrioventricular node and spreads to more distant points (Amat-y-Leon *et al.*, 1976; Gallagher *et al.*, 1977). The relative timing of atrial activation at different sites is determined by intra-atrial conduction times to the sites of recording. When two or more ventriculoatrial pathways are present the atrial activation patterns depends on the relative contribution to atrial depolarisation by each conduction pathway. The presence of two or more concealed ventriculoatrial accessory pathways may make interpretation difficult especially if these pathways have similar characteristics and location. Several determinants may interact to produce any given activation pattern (Fig. 7). Though we have seen 4 basic patterns of ventriculoatrial conduction in patients with concealed accessory pathways, other varieties may occur. For example, atrial fusion at all coupling intervals of V_2 was reported by Sung *et al.* (1977a) in 3 of 29 patients with type A Wolff-Parkinson-White syndrome. The presence of intra-atrial or intraventricular delay or gaps in ventriculoatrial conduction in the normal pathway (Akhtar *et al.*, 1974) or accessory pathway may result in variations of basic patterns. In addition, the basic pattern may be modified by changes in the ventricular driving rate.

Retrograde atrial activation patterns may be important in the diagnosis and location of concealed accessory pathways especially in those patients in whom it may not be possible to initiate tachycardia at electrophysiological study. It is clear that constancy of the ventriculoatrial interval is, by itself, an inadequate criterion for the diagnosis of concealed ventriculoatrial pathways. If the V-RA interval is considered, a progressive increase was shown in 8 patients (group 3 and group 4) and a sudden increase in 2 patients one of whom had intra-atrial delay. In the absence of left atrial recordings these V-RA patterns may have been reasonably attributed to conduction in the normal pathway alone. For this reason, it is important that atrial activity should be recorded from sites closely adjacent to both normal and accessory pathways.

Recently it has been re-emphasised that concealed accessory pathways may in fact be the anatomical substrate for tachycardias previously diagnosed as depending on intranodal re-entry (Barold and Coumel, 1977; Sung *et al.*, 1977b; Pritchett *et al.*, 1978). The distinction between these mechanisms is clearly of therapeutic importance particularly if surgical intervention is considered.

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